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Title: One-year risks of cancers associated with CO 19 vaccination: a large population-based cohort study in South Korea. **Authors:** Hongjin Kim, Minho Kim, Mayong Choi, and Uni Chun.

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Part I: The Horizon of Long-Term Monitoring

Speaker 1: Hello and welcome back. We are—uh—we are diving deep today. And when I say "deep," I mean we are wading into a data set that is honestly just staggering in its sheer scale. We're talking about millions upon millions of people.

But before we get to the numbers—and trust me, the numbers are going to demand a lot of our attention today—I kind of want to set the scene. For the last few years, the conversation around vaccines, specifically the CO 19 vaccines, has, you know, mostly focused on the immediate stuff, right? To short term.

Speaker 2: Yeah, you know the drill. The sore arm, the fever, the fatigue—maybe those rare cases of myocarditis that pop up within a few weeks. It's all very acute. It's all very "right now".

Speaker 1: Exactly. It's the acute phase monitoring. And that's usually where the safety protocols start and stop for the general public's attention span. We look for immediate reactions, we log them, and well, we move on, right? But this paper, this paper is doing something fundamentally different. It's looking at the horizon. It's looking at the one-year mark.

Speaker 2: Well, a much longer view.

Speaker 1: And it's asking a question that I think a lot of people have been afraid to ask, or maybe just assumed we didn't have the data for yet. The question is: **Is there a link—an epidemiological link—between these vaccines and cancer?** It is the heavy question. And to be clear, asking that question isn't about fear-mongering; it is—uh—it's about doing the necessary science. We're now far enough out from the initial global rollout to start gathering this kind of retrospective data.

Speaker 2: Yeah. And what makes this specific study by Kim and colleagues so pivotal is, as you hinted, the scale. This isn't a survey of 500 people in a local clinic. No, this is a massive population-based cohort study using the National Health Insurance Service database in South Korea. I want to get into those numbers in a second because they are just—they're mind-boggling.

But first, let's tackle the premise, because for a lot of people listening, the idea that a vaccine for a respiratory virus could trigger cancer might sound like a massive leap.

Part II: The Biological Hypothesis

Speaker 1: It sounds, well, biologically distant. Why would researchers even suspect this link? Why burn the calories to run a study on millions of people if there isn't a plausible mechanism?

Speaker 2: It's a completely valid question. And to understand the hypothesis, we have to look at the virus itself first. The authors of this paper ground their research in the biological context of SARS-CoV-2. We know that certain viruses have what we call **oncogenic potential**.

Speaker 1: "Oncogenic" meaning cancer-causing—like they have the ability to somehow flip a switch in our cells.

Speaker 2: Precisely. Think about HPV (human papillomavirus) and its established link to cervical cancer, or, you know, the Epstein-Barr virus and its connection to certain lymphomas. Right. We have established precedents for viruses hijacking cellular machinery in a way that leads to uncontrolled cell growth.

The authors point out that SARS-CoV-2 shares some of these potential mechanisms. For one, it interacts with the renin-angiotensin-aldosterone system, or **RAAS**.

Speaker 1: Okay, let's unpack that. The renin-angiotensin-aldosterone system. That sounds like a mouthful. I usually think of that in terms of blood pressure, right?

Speaker 2: Usually, yes, that's its day job. It regulates blood pressure and fluid balance, but it also acts as a major regulator for cell growth and—uh—and inflammation. And here's the key mechanism the authors are worried about: the virus, and by extension the vaccine, introduces the spike protein.

Speaker 1: Mhm.

Speaker 2: We all know the spike protein targets the ACE2 receptor to enter cells.

Speaker 1: ACE2—that's the doorway, the key in the lock.

Speaker 2: It is the doorway, but it's also a brake pedal. ACE2 normally regulates that RAAS system we just mentioned. It keeps inflammation in check. When the spike protein binds to ACE2, it effectively breaks the valve. It downregulates ACE2.

Speaker 1: So, it takes the regulator offline.

Speaker 2: Exactly. And when you lose that regulation, you don't just get blood pressure issues, you get a cascade of pro-inflammatory signals. It's like the system goes into overdrive.

Speaker 1: So, by blocking the regulator, you're essentially stepping on the gas pedal for inflammation.

Speaker 2: Exactly. And chronic—or in this case, acute—**hyperinflammation** is a known driver of cancer. I mean, this is well established. It damages DNA. It disrupts the body's ability to repair that DNA, and it creates a protumorigenic environment—an environment where cancer can thrive.

Speaker 1: And the authors explicitly mention this concept of "COVID-19 vaccine-induced hyperinflammation".

Speaker 2: They do.

Speaker 1: So the hypothesis then is that the vaccine, by introducing the spike protein to generate immunity, might inadvertently be triggering this exact same inflammatory cascade that we see with the virus.

Speaker 2: That is the working theory. The authors hypothesized that because the vaccines share this specific structure—the spike protein—with the virus, they might also share some of those risks. They mention other potential pathways too, like—uh—interactions with tumor suppressor genes like P-53 and even the possibility of reactivating dormant viruses that are already in our systems.

Speaker 1: Like Epstein-Barr, which you mentioned.

Speaker 2: Exactly. There's a complex web, but the core idea is that the inflammatory shock might be enough to wake up "sleeping giants," so to speak.

Part III: Methodology and Statistical Twins

Speaker 1: "Sleeping giants"—that is a terrifying image. But it brings us to the methodology, because you can have all the theories in the world, but you need data to prove it.

Speaker 2: And the authors state clearly that until this study, real-world data were insufficient. They set out to close that data gap and, well, they closed it with a sledgehammer of the data set. Let's look at section two here: the study design. I mentioned millions earlier; the total pool they started with was **8,427,849 individuals**.

Speaker 1: Just let that number sink in. 8.4 million people.

Speaker 2: That is essentially the entire population of Seoul.

Speaker 1: It's a remarkable data set. But in epidemiology, raw numbers aren't enough. In fact, raw numbers can be dangerous if you don't treat them with respect. You need to compare apples to apples.

Speaker 2: What do you mean?

Speaker 1: Well, if you just compare everyone who got vaccinated to everyone who didn't, you run into massive problems, right? Because the people who got vaccinated might be different. Maybe

they're older and more health-conscious. Maybe they're healthcare workers, or maybe the unvaccinated people were already too sick to get the shot in the first place.

Speaker 2: Exactly. All of that. That's called selection bias or sometimes the "healthy user bias". To eliminate this, the researchers used a really powerful technique called **Propensity Score Matching**, or PSM.

Speaker 1: Okay, I want to pause on propensity score matching. It sounds like academic jargon, but it's actually the referee in this game. If we don't understand this, we can't trust the results. What is PSM actually doing?

Speaker 2: Yeah, let's break it down. Imagine you have a vaccinated 45-year-old male living in Seoul who makes a middle-class income, has a history of mild hypertension, and, you know, visits the doctor twice a year. To make a fair comparison, you cannot compare him to a healthy 20-year-old female who never goes to the doctor. It's a meaningless comparison. You need to find his "statistical twin" in the unvaccinated group.

Speaker 1: You need to find another 45-year-old male with hypertension and the same income who didn't get the shot.

Speaker 2: Precisely. The researchers assigned every single person in that 8 million-person pool a propensity score based on a whole list of factors, right? Age, sex, insurance premium levels (which is a good proxy for income), region of residence, and the Charlson Comorbidity Index, which is a standardized way to measure a person's underlying health issues.

Speaker 1: So they build a complete profile for every single person.

Speaker 2: A very detailed one. Then they matched them. They did this at a ratio of 1 to 4. For every one unvaccinated person, they found four vaccinated twins who looked almost identical on paper in all those key aspects.

Speaker 1: And if they couldn't find a good match for someone?

Speaker 2: They threw the data out. Yes, they sacrificed volume for accuracy. This is a sign of a really rigorous study. They're prioritizing the integrity of the comparison over just having bigger numbers. And this rigorous process whittled that 8 million down to a final cohort for analysis.

Speaker 1: And that final cohort was still what—about 595,000 unvaccinated individuals compared against roughly 2.38 million vaccinated individuals?

Speaker 2: That is still a massive study. Even after throwing out all the unmatched data, you have nearly **3 million people** in the final analysis. That is bigger than the population of some countries.

Speaker 1: And that statistical power is what allows them to detect signals that smaller studies would miss. If a risk increases by, say, 20% or 30% for a specific cancer, you might not see it in a clinical trial of 30,000 people. It just gets lost in the statistical number.

Speaker 2: It's too faint.

Speaker 1: Exactly. But in a group of 3 million, that signal shines like a beacon. It allows us to see these subtle but incredibly important shifts in population health. And the timeline here is crucial too, right? We are looking at one-year risks. This covers the period from 2021 to 2023. So, we are looking at the cumulative incidence of cancer appearing one year post-vaccination.

Speaker 2: Correct. From the date of their first shot, the clock starts ticking for one year.

Part IV: Acceleration vs. Initiation

Speaker 1: Wait, I have to stop you there. We're talking about a one-year risk. Biologically, that doesn't track with how we usually talk about this disease. Cancer is a "slow burn".

Speaker 2: It is.

Speaker 1: It takes years, sometimes decades, for a DNA mutation to turn into a tumor you can actually see on a CT scan. How can a vaccine cause a jump in cancer rates in just 12 months? Are we saying it planted the seed, or are we saying it poured gasoline on a fire that was already there?

Speaker 2: That is the million-dollar question, and it's something the authors tackle. You're absolutely right. It is highly unlikely—bordering on impossible—that the vaccine initiated a brand-new cancer from scratch, from the very first mutation to a detectable tumor, in just 12 months. That is not how oncogenesis typically works.

Speaker 1: So what are we seeing then? Is it just misdiagnosis or something else?

Speaker 2: The leading hypothesis is not initiation but **progression or acceleration**. The idea is that many of us, as we get older, have micro-cancers—tiny, dormant clusters of malignant cells that our immune system usually keeps in check.

Speaker 1: Our bodies are constantly fighting off these little rogue cells.

Speaker 2: Constantly. Your NK cells (your natural killer cells) and your T-cells are on patrol handling them. But if the vaccine induces a state of hyperinflammation or even temporary immune dysregulation, it might disrupt that surveillance. It's like the security guards are all off to deal with a riot at the front gate, and in the chaos, prisoners escape from their cells.

Speaker 1: Wow.

Speaker 2: So, it's not planting the seed. It's watering the soil and removing the gardener at the same time, allowing what was already there but dormant to grow and become clinically detectable.

Part V: The "Big Six" Findings

Speaker 1: That is a chilling analogy. So, we are looking at existing problems being amplified. Okay, let's get to the part everyone is waiting for: the numbers. The study looked at a whole laundry list of cancers, but they found significant increases in risk for six specific types.

Speaker 2: Yes, the "big six," if you will.

Speaker 1: And before we list them, we need to explain the scoreboard. We're going to be talking about **Hazard Ratios**, or HR. Can you explain what that is?

Speaker 2: Of course. An HR of 1.0 is the baseline. It means there is absolutely no difference in risk between the vaccinated and the unvaccinated groups. It's a wash.

Speaker 1: Okay, 1.0 is neutral.

Speaker 2: Right. If the HR is 1.5, that indicates a 50% increased risk in the vaccinated group compared to the unvaccinated group. If it's 2.0, that's double the risk. And if it were, say, 0.5—that would mean a 50% decrease in risk.

Speaker 1: Got it. Okay, so let's walk through these six findings slowly, because each one represents a significant health burden. The first one—and the one with the highest hazard ratio—was **prostate cancer**.

Speaker 2: This is the strongest signal in the entire data set. The hazard ratio for prostate cancer was **1.687**.

Speaker 1: 1.687—that is—that's nearly a 70% increase in risk.

Speaker 2: It is—to be precise—a **68.7% increase** in the risk of being diagnosed with prostate cancer within one year of vaccination compared to an unvaccinated but otherwise similar person.

Speaker 1: I see these numbers in the table here. The confidence interval is 1.348 to 2.111. Translate that for me. Is there any wiggle room here? Could this just be random noise?

Speaker 2: That's a great question. The confidence interval is like the margin of error in a political poll. It gives us a range of plausible values.

Speaker 1: Okay.

Speaker 2: If that range crossed the number 1.0—if it was, say, 0.9 to 1.8—it would mean there's a statistical chance the vaccine did nothing at all or was even protective.

Speaker 1: The result wouldn't be significant.

Speaker 2: Correct. But here, the entire range is well above 1.0. The floor of our estimate—the absolute best-case scenario in the math—is still a 34.8% increase in risk. The ceiling is over a 111% increase—more than double. The truth lies somewhere in between, likely around that 68% mark. But the math is telling us with a very high degree of confidence: this is not a coincidence.

Speaker 1: A 68% increase in prostate cancer—that is massive. We are talking about one of the most common cancers in men essentially surging within a year.

Speaker 2: It's a profound finding, and as we'll get into a bit later, this was a signal driven heavily by the older population. But the sheer magnitude of that HR 1.687 is something you rarely see in these types of broad observational studies unless there is a genuine biological interaction happening.

Speaker 1: A huge red flag.

Speaker 2: It is.

Speaker 1: So we have prostate cancer screaming at us from the data. But what about the rest of the body? Because if this is a systemic inflammation issue—this hyperinflammation theory—we should see it pop up elsewhere, right?

Speaker 2: And we do. It's not an isolated finding, but it appears most aggressively in organs that are, well, highly vascular or heavily regulated by hormones. Take the **lungs**, for instance. This was number two on the list. The hazard ratio for **lung cancer was 1.533**.

Speaker 1: 1.533—that represents a more than 50% increase in the risk of being diagnosed with lung cancer within one year of vaccination.

Speaker 2: A 53.3% increase to be exact. And remember, lung cancer is a major killer. Globally, a 50-plus percent increase in incidence, even over a short period, is a public health disaster in terms of raw numbers.

Speaker 1: And the biological link here seems more direct, I guess—it's more intuitive.

Speaker 2: I think so. The lungs are the primary site of infection for the virus itself, and they are the site where the inflammatory response to respiratory distress is most acute. It seems plausible that the vaccine, by mimicking that viral presence, might be triggering a similar inflammatory environment in the lung tissue that could accelerate tumor growth.

Speaker 1: Okay, so that's two major cancers. What was number three on the list?

Speaker 2: Number three: **thyroid cancer**. The hazard ratio here was **1.351**.

Speaker 1: So about a 35% increase in risk. This one feels different to me. The thyroid isn't a filtering organ like the lungs or the liver; it's a hormone gland.

Speaker 2: Exactly. And that is why it's so interesting and supports the broader theory of immune dysregulation. Thyroid cancer often involves the endocrine system and inflammatory pathways. The thyroid is incredibly sensitive to immune signaling. We already know about autoimmune thyroiditis—a condition where the immune system mistakenly attacks the thyroid.

Speaker 1: Like Hashimoto's disease.

Speaker 2: Precisely. The authors suggest that the vaccine-induced immune response might be tipping the scales in thyroid tissue, promoting tumorigenesis in individuals who might already have been susceptible.

Speaker 1: Okay. Number four: **gastric cancer** (stomach cancer).

Speaker 2: Hazard ratio **1.335**. Again, roughly a one-third increase in risk—a 33.5% jump.

Speaker 1: This one is particularly insidious because gastric cancer can be very hard to detect early. It's often asymptomatic until it's quite advanced.

Speaker 2: It is. And as we will see later when we talk about boosters, this particular cancer signals very, very strongly. It's one to watch.

Speaker 1: And number five?

Speaker 2: Number five: **colorectal cancer**. The HR here was **1.283**.

Speaker 1: A 28% increase. This is another one of the most common cancers. We screen for this all the time with colonoscopies starting at age 45 now, right? And a nearly 30% increase translates to a very large number of actual cases in a population of millions.

Speaker 2: And the biological link there: the gut is a major immune organ. People forget that a huge portion of your immune system lives in your gut lining—the GALT, or gut-associated lymphoid tissue. So if you have systemic hyperinflammation, the colon is often on the front lines. We see it with inflammatory bowel diseases like Crohn's and colitis, which themselves are risk factors for colon cancer. It follows a similar logic.

Speaker 1: And finally, number six: **breast cancer**.

Speaker 2: The hazard ratio was **1.197**, effectively a **20% increase**. Now, 20% might sound low compared to the nearly 70% we saw with prostate cancer.

Speaker 1: In relative terms, yes, the percentage jump is smaller.

Speaker 2: But you have to look at the prevalence of the disease itself.

Speaker 1: Ah, okay.

Speaker 2: Breast cancer is the most common cancer in women globally. A 20% bump in the incidence of the most common cancer is epidemiologically enormous. That represents thousands and thousands of women in this data set alone. A small percentage of a very big number is still a very big number.

Speaker 1: So just to recap for listeners: we have prostate, lung, thyroid, gastric, colorectal, and breast cancer. These aren't obscure, rare diseases; these are the big ones. These are the cancers that everyone knows someone who has battled.

Speaker 2: Precisely. And the consistency of the signal across these major organ systems—lungs, gut, hormones, reproductive organs—is what concerns the authors. It's not just one freak signal in the pinky toe; it's systemic.

However, we also have to appreciate the nuance in the data. And this is important: the study didn't find that everything went up. If this was just, you know, a poison, you'd expect every cell in the body to have a negative reaction, right? But that's not what happened.

Speaker 1: No.

Speaker 2: And this is where the science gets really fascinating and complicated. There were exceptions, and there were significant ones. When they looked at hematologic cancers (cancers of the blood), the signal was actually reversed.

Speaker 1: "Reversed?" What do you mean reversed?

Speaker 2: The risk went down. For leukemia, the hazard ratio was **0.56**. For myeloma, it was **0.58**.

Speaker 1: Wait, let me do the math. That means the vaccinated group had roughly a **44% lower risk of leukemia** and a **42% lower risk of myeloma**?

Speaker 2: According to this data, in this one-year window, yes.

Speaker 1: How is that possible? I mean, how can the same shot cause a nearly 70% increase in prostate cancer and a 40% decrease in leukemia? That seems paradoxical.

Speaker 2: It does, but it also highlights the complexity of the biological interaction. It's not a simple on-off switch. It suggests that the immune modulation caused by the vaccine—that massive ramp-up of T-cells and antibodies—might actually be protective against certain blood cancers.

Speaker 1: Wow.

Speaker 2: Well, leukemia and myeloma are cancers of the blood and bone marrow—the very home of the immune system. It's possible that the "high alert" state of the immune system post-vaccination actually helps to find and clear out early-stage leukemic cells before they can establish themselves. The immune surveillance for those specific cancers might have been enhanced.

Speaker 1: So, it's like the immune system is being tuned, but in a very specific way. It's tuned up to fight blood cancer, but that same tuning—that same inflammation—is causing collateral damage in the solid organs like the prostate and lungs.

Speaker 2: That is a very plausible interpretation of this data. It's a trade-off. The immune system is a powerful, blunt instrument. When you activate it this strongly, it can have both beneficial and detrimental effects simultaneously in different parts of the body.

Part VII: Vaccine Types—mRNA vs. cDNA

Speaker 1: That's incredible. It tells us that vaccination isn't a simple "good" or "bad" switch; it's a complex biological input that ripples through different systems in very different ways.

Speaker 2: Exactly.

Speaker 1: Now, another layer of this onion is the vaccine type itself. In South Korea, like many places, they didn't just use one brand. They had the mRNA vaccines (Pfizer, Moderna) and the cDNA or viral vector vaccines (like AstraZeneca and Janssen).

Speaker 2: Right. And this is a crucial distinction because these are fundamentally different technologies for achieving the same goal.

Speaker 1: Explain that.

Speaker 2: Well, mRNA uses a little bubble of fat—a lipid nanoparticle—to deliver the genetic instructions for the spike protein directly into your cells. The cDNA technology uses a modified, harmless chimpanzee adenovirus (basically a cold virus that can't replicate) to carry those same instructions.

Speaker 1: So, a different delivery truck for the same package.

Speaker 2: A very different delivery truck, and that seems to matter. The study found a difference between these technologies. Did it matter which shot you got? The answer is yes.

Speaker 1: Okay, so the authors broke this down in detail. Let's start with the cDNA ones—the viral vectors, AstraZeneca.

Speaker 2: The cDNA vaccines were associated with increased risks in **five of the six categories** we just discussed.

Speaker 1: Five of the six?

Speaker 2: Specifically: thyroid, gastric, colorectal, lung, and prostate cancers.

Speaker 1: So almost a full sweep of the bad list. The only one not on that list was breast cancer.

Speaker 2: Correct. For the viral vector group, the breast cancer signal was not statistically significant. So it seemed to have a broader oncogenic footprint in this study, covering the gut, lungs, and prostate quite strongly. Now, what about the mRNA vaccines? These are the ones most people in the US and Europe received—Pfizer and Moderna.

Speaker 2: The mRNA vaccines were linked to increased risks in **four categories**.

Speaker 1: Okay.

Speaker 2: Those were thyroid, colorectal, lung, and breast cancers.

Speaker 1: Okay, let's compare those lists. It looks like gastric and prostate risks were more specific to the viral vector shots.

Speaker 2: Based on this cohort, yes. The signal for gastric and prostate cancer was much stronger and statistically significant in the cDNA group, but it didn't reach significance in the mRNA group alone.

Speaker 1: But breast cancer?

Speaker 2: That was the opposite. The breast cancer risk appeared to be more specific to the mRNA group. It was significant there, but not in the cDNA group.

Speaker 1: And it looks like lung, thyroid, and colorectal risks were elevated across the board, regardless of the technology used.

Speaker 2: Exactly. That suggests those particular risks might be tied directly to the spike protein itself (which both vaccines produce) or just the general inflammatory response that any vaccine causes.

Speaker 1: But the difference is the breast cancer signal in mRNA versus the prostate and gastric signal in cDNA. That suggests that the delivery mechanism also plays a role.

Speaker 2: It has to. Maybe the lipid nanoparticles distribute differently in the body than the viral vectors. Maybe they accumulate in breast tissue or associated lymph nodes. Maybe the viral vector has a specific affinity for the prostate or the gut lining. We don't have the molecular studies to confirm *why* yet, but the data clearly points to a difference.

Speaker 1: And there's a third group in this analysis, right? The heterologous group. That means people who mixed and matched.

Speaker 2: Exactly. People who maybe got AstraZeneca for dose 1 and Pfizer for dose 2. This is a common strategy in some countries.

Speaker 1: What did they find there?

Speaker 2: The study found that this mix-and-match group showed specific increased risks for **thyroid and breast cancers**.

Speaker 1: It's just—it's so specific. It really highlights that vaccination isn't a single variable; it's a spectrum. If you're a clinician, knowing that an mRNA vaccine might carry a specific risk profile for breast cancer that a cDNA vaccine might not carry to the same extent—that is incredibly actionable information for personalized medicine.

Part VIII: Demographics—Age and Sex

Speaker 2: It is. And it brings us to the next variable, which is maybe the most important one: **who you are**. We're not just machines receiving a shot; we're talking about age and sex, because biology isn't the same for a 20-year-old woman and an 80-year-old man.

Speaker 1: Absolutely. Stratification is key. Let's look at the sex differences first—men versus women.

Speaker 2: The study found very distinct vulnerabilities. For males, the vaccinated group was significantly more vulnerable to **gastric cancer and lung cancer**.

Speaker 1: Gastric and lung for men. And for females?

Speaker 2: Vaccinated females were more susceptible to **thyroid and colorectal cancers**.

Speaker 1: That thyroid signal for women—that seems consistent with what we know about thyroid issues generally being more common in women, doesn't it?

Speaker 2: It does, very much so. Autoimmune thyroid disease is about eight times more prevalent in women. It suggests the vaccine might be exacerbating a pre-existing biological predisposition in that demographic. It's taking a system that is already, for whatever reason, on the edge, and perhaps pushing it over.

Speaker 1: Okay, so that's sex. Now, what about age? This is the part of the paper that actually surprised me the most. You usually assume older people are more at risk for everything. If you tell me cancer rates went up, I immediately assume you mean in the 80-year-olds.

Speaker 2: And in terms of raw numbers, of course, that's true. Cancer is fundamentally a disease of aging; you will always see more total cases of cancer in the elderly.

Speaker 1: And that's not what the hazard ratio showed?

Speaker 2: No. When we look at the relative risk elevation—the hazard ratio which compares the vaccinated to their unvaccinated peers—the story flips.

Speaker 1: Yeah.

Speaker 2: The authors note, and I'm quoting here, that "the vaccination associated cancer risk was likely more elevated among individuals aged <65 years".

Speaker 1: So, the younger group saw a steeper increase in risk relative to their unvaccinated peers.

Speaker 2: Exactly. Specifically, the population under 65 was more vulnerable to thyroid and breast cancers.

Speaker 1: Why is that more alarming, in a way?

Speaker 2: Because if a 30-year-old gets breast cancer, it feels like an anomaly—the baseline risk for their age group is very, very low. So if you see a significant spike in that group, it suggests a strong external driver is at play. If an 80-year-old gets cancer, it's tragic, but it's biologically less surprising. Finding a sharp increase in cancer risk in a younger cohort indicates that the vaccine might be disrupting biological systems that should be healthy and robust.

Speaker 1: Right. This is disrupting the prime of life. But there was one major exception to this "younger is more at risk" trend.

Speaker 2: Correct. We have to go back to the prostate.

Speaker 1: Yes, prostate cancer.

Speaker 2: This was the outlier. The older population—specifically those **75 and older**—were significantly more susceptible to the vaccine-associated increase in prostate cancer. And given that the hazard ratio for prostate cancer was the highest in the entire study at 1.687, this is a very specific, very serious signal for the elderly male population.

Part IX: Boosters and the Pancreatic Signal

Speaker 1: It really paints a picture of customized risk. It's not "everyone is in danger". It's: older men need to watch the prostate, younger women need to watch the thyroid and breasts.

Speaker 2: Which is exactly why broad epidemiological studies like this are so valuable. They allow us to move from the simplistic question, "Is it safe?" to the much more intelligent questions of "Who is it safest for?" and "What specifically should we be monitoring, and in whom?"

Speaker 1: Okay, and that brings us to boosters. The study didn't just look at the primary series of one or two shots; it looked at the effect of getting those additional doses.

Speaker 2: Right. This is section six of our dive, and honestly, it contains some of the most striking data in the whole paper. The authors state that boosters "substantially affected the risk profile for specific cancers".

Speaker 1: Meaning the risk didn't stay static; it changed with re-exposure to the vaccine.

Speaker 2: Correct. The whole concept of a booster is re-exposure to the antigen to spike your antibody levels back up. But if the antigen (the spike protein) or the immune response it generates is what's driving this oncogenic risk, then re-exposure could logically compound that risk.

Speaker 1: More fuel on the fire.

Speaker 2: Potentially. And the study highlights a significant higher risk of gastric cancer in vaccinated individuals. And the authors explicitly write—and this is another quote—"clinicians should prioritize monitoring the risk of gastric cancer in relation to CO 19 booster doses".

Speaker 1: That is a direct instruction to doctors: "Prioritize monitoring". They aren't mincing words there. That's a clinical recommendation based on their findings.

Speaker 2: They are not. But there is a statistic buried in table one of the source material that we absolutely have to discuss. It wasn't in the abstract summary; it wasn't in the "big six" list; but when you dig into the data tables, it stops you in your tracks. It's about **pancreatic cancer**.

Speaker 1: Oh, I saw this. This was the moment I put my coffee down and just stared at the page.

Speaker 2: Pancreatic cancer is one of the most lethal malignancies we deal with. It is silent, it's fast, and it is incredibly hard to treat. The survival rates are notoriously low. In the primary analysis of just vaccinated versus unvaccinated, pancreatic cancer didn't make the "big six".

Speaker 1: Right. The signal wasn't statistically significant overall. It was a little elevated but not enough to be conclusive.

Speaker 2: But when the researchers analyzed the booster group—specifically people who got the extra shots—they found a hazard ratio for **pancreatic cancer of 2.25**.

Speaker 1: 2.25? That is more than double the risk. That is a **125% increase in risk**.

Speaker 2: It is. The 95% confidence interval is 1.44 to 3.50, and the p-value is less than 0.001.

Speaker 1: For those who don't speak statistics, what does a p-value of less than 0.001 mean?

Speaker 2: It means the probability of this result happening by random chance is less than one in a thousand. In statistical terms, that is a flashing red siren. It's a highly, highly significant finding.

Speaker 1: So, let me get this straight. You get the first two shots and maybe your pancreatic risk is baseline or slightly elevated, but you get the booster and suddenly the risk jumps to over double that of an unvaccinated person?

Speaker 2: That is what this data from South Korea indicates. It implies that dose accumulation—the repeated stimulation of the immune system—might be the trigger for this specific pathology. Maybe the pancreas can handle one or two hits of inflammation, but a third hit is what pushes it over the edge into detectable disease.

An HR of 2.25 is massive. Most epidemiological studies get excited about an HR of 1.1 or 1.2. A 2.25 for something as deadly as pancreatic cancer is a finding that demands immediate, focused follow-up research.

Speaker 1: It's terrifying, frankly. Pancreatic cancer isn't something you usually catch early with routine screening. If boosters are more than doubling that risk, that is a catastrophic public health signal hidden in the data.

Speaker 2: It explains why the authors are so cautious but clear in their conclusion. They aren't trying to cause a panic; they are simply presenting the data and saying, "Look at this".

Speaker 1: They are saying the "one-size-fits-all" strategy is flawed in the face of this data. You can't just tell everyone to get boosted indefinitely without considering these potential risks.

Speaker 2: Exactly. If you have a signal this strong for pancreatic cancer with boosters, or prostate cancer in the elderly, or breast cancer in young women receiving mRNA, you cannot ethically ignore it.

Speaker 1: So, let's try to wrap our heads around the implications here. We have a massive study—millions of people. We have solid methodology with propensity score matching, and we have these clear, statistically significant signals for six major cancers, plus this alarming pancreatic cancer signal with boosters. What is the final takeaway?

Speaker 2: The takeaway is summarized very well by the authors' own conclusion. They state: "Further research is needed to determine whether specific vaccination strategies may be optimal". That is academic-speak for "We need to change how we do this," right? It's a polite way of saying the current strategy might not be the best one. We need to tailor the strategy.

Speaker 1: We need to acknowledge that the benefit-risk ratio isn't a flatline. It's not the same for everyone. It curves. It changes based on your age, your sex, which specific vaccine you use, and—critically—how many doses you take. The authors clearly believe that the current blanket approach needs re-evaluation. They specifically mentioned the need to monitor gastric risk in men and thyroid risk in women.

Speaker 2: And they also circle back to the mechanism at the end. This is an observational study; it sees the smoke, but we still need to find the fire, right? Correlation is not causation—even with a hazard ratio of 2.25, right? It's a very, very strong correlation, but we need to confirm *how* this is happening at a molecular level.

The authors call for molecular studies to understand the hyperinflammation mechanism. Is it the spike protein directly interacting with p-53 tumor suppressor genes? Is it the disruption of the innate immune system's ability to clear cancer cells? Is it the reactivation of dormant viruses like EBV, as they hinted at in the intro? We don't know the precise molecular *how* yet, but the epidemiological *what* is now on the table.

Speaker 1: And "on the table" means it's in the peer-reviewed scientific literature. It's published in *Biomarker Research*. It's not a conspiracy theory on a forum board; it's data.

Speaker 2: Correct. And that gives it weight. It gives other researchers around the world permission—and a roadmap—to look for these same signals in their own national health databases: in the US, the UK, Europe, everywhere.

Speaker 1: When I look at this entire paper—the prostate risk in the elderly, the thyroid risk in the young, the pancreatic signal in the boosted—it feels like the end of the one-size-fits-all era of public health, or at least it should be.

Speaker 2: It has to be. This study is essentially a roadmap for what we could call **precision public health**. The authors are saying that if we want to maintain trust and maximize safety, we have to stop treating the entire population as a monolith. If you are a 30-year-old woman, your risk calculation for both COVID and the vaccine is fundamentally different from an 80-year-old man's. And ignoring that biological reality doesn't make the vaccine safer; it just makes our monitoring blinder.

Speaker 1: And that brings us to the most uncomfortable question—the one that lingers after you read this. We're looking at Year One—a one-year risk. If this mechanism is real—if this inflammation is accelerating tumor growth—what does the chart look like at Year Five or Year Ten?

Speaker 2: That is a provocative thought, isn't it? In oncology, one year is a blink of an eye. Usually, we measure the effects of carcinogens over years or decades. If we are seeing signals this strong now—a 68% increase here, a 125% increase there—so early on, it implies a powerful acceleration of disease.

It opens the door to the possibility of long-term vigilance that, frankly, our public health systems haven't fully grappled with yet. We might be looking at a long tail of oncological consequences that will require resources, screening, and a lot of honesty to manage in the coming years.

Speaker 1: It's a sobering thought, but I suppose knowledge is better than ignorance. At least with a study like this, we know where to start looking. We know to check the thyroids, check the prostates, to ask patients about gastric symptoms after boosters. We aren't flying completely blind anymore.

Speaker 2: Exactly. The data is the flashlight. It might show us things we don't want to see in the dark, but it's always better than stumbling around without it.

Speaker 1: Well, on that note—a heavy note, but a necessary one—we are going to wrap up this deep dive. I hope this helped you understand the numbers behind the headlines, or perhaps the headlines that *should* be out there but aren't. Thank you.

Speaker 2: Thank you for listening to another session of the Notebooklm.news series. You can find the full archive of all my podcasts at notebooklm.news.